

**Editorial:****METAL TOXICITY**

J.D. Stewart

Leibniz Research Centre for Working Environment and Human Factors (IfADo), Ardeystrasse 67, 44139 Dortmund, Germany

E-mail: [stewart@ifado.de](mailto:stewart@ifado.de), Telephone: +49 231-1084-406, Fax: +49 231-1084-403

Metal Toxicity is one of the cutting-edge topics of our partner journal Archives of Toxicology. Unlike many other compounds metals are not degraded facilitating the accumulation of toxic concentrations (Beyersmann and Hartwig, 2008). Mechanisms of metal toxicity include oxidative stress induction, DNA repair inhibition, modification of cell proliferation and cadherin interference. Due to the multitude of mechanisms involved and the target structures, metal toxicity represents an extremely complex field of research. To give our readers an overview of recent publications on metal toxicity we have summarised their key messages in Table 1.

**Table 1:** Recent results in research on **metal toxicity**

Key message	Reference
Potassium dichromate inhibits brush border membrane enzymes and causes oxidative stress in rat intestine.	Arivarasu et al., 2008
This article reviews the toxic mechanisms of aluminium and lead in brain: membrane biophysics, altered cell signalling and the impairment of neurotransmission are highlighted.	Verstraeten et al., 2008
The paper describes mechanisms on how methyl-mercury suppresses arginase I activity: covalent modification of MeHg and leakage of Mn ions from the active site.	Kanda et al., 2008a
Cadmium, cobalt and lead cause characteristic alterations of gene expression patterns in cultivated human bronchial epithelial cells.	Glahn et al., 2008
No interaction between in vivo relevant concentrations of arsenic and malathion was observed in a rat in vivo study.	Naraharisetti et al., 2008
This is a comprehensive review about the molecular mechanisms of carcinogenic metals.	Beyersmann and Hartwig, 2008
A meta-analysis shows that lead may reduce nerve conduction velocity. The lowest lead blood concentration at which a relationship could be detected was 33.0 microg/dl.	Krieg et al., 2008
Cadmium alters transferrin and hepcidin expression in fish.	Chen et al., 2008
Copper gluconate may act as a rat liver carcinogen as evidenced by a medium-term liver carcinogenicity protocol.	Abe et al., 2008
Diphenylarsinic acid is a product of degradation of arsenic-containing chemical weapons and was detected in well water in Japan. This is a pharmacokinetic study in cynomolgus monkeys describing distribution and excretion of arsenic after repeated administration of diphenylarsinic acid.	Kobayashi et al., 2008
Lead acetate administered into the yolk sac causes developmental neurotoxicity in chicks.	Müller et al., 2008

Key message	Reference
Lead induced foetal developmental toxicity in mice is enhanced by meso-2,3-dimercaptosuccinic acid.	Yu et al., 2008
Methyl mercury and mercuric sulfide modify pentobarbital induced hypnotic tolerance.	Chuu et al., 2008
Cisplatin-induced nephrotoxicity in rats is attenuated by the phytoalexin resveratrol.	Do Amaral et al., 2008
Arsenic-induced oxidative myocardial injury in mice is ameliorated by arjunolic acid.	Manna et al., 2008
Inorganic mercury accumulates in the proximal tubules of the kidney and causes apoptosis. Apoptosis induction in this context is antagonized by overexpression of arginase II.	Kanda et al., 2008b
An optimum combination of micronutrients (calcium and ascorbic acid) with 2,3-dimercaptosuccinic acid for the treatment of lead-intoxicated mice was established.	Liao et al., 2008
Vitamin D receptor gene variants are associated with circulating concentrations of lead in exposed humans.	Rezende et al., 2008

## REFERENCES

Abe M, Usuda K, Hayashi S, Ogawa I, Furukawa S, Igarashi M, Nakae D. Carcinogenic risk of copper gluconate evaluated by a rat medium-term liver carcinogenicity bioassay protocol. Arch Toxicol 2008;82:563-71.

Arivarasu NA, Fatima S, Mahmood R. Oral administration of potassium dichromate inhibits brush border membrane enzymes and alters anti-oxidant status of rat intestine. Arch Toxicol 2008;82:951-8.

Beyersmann D, Hartwig A. Carcinogenic metal compounds: recent insight into molecular and cellular mechanisms. Arch Toxicol 2008;82:493-512.

Chen J, Shi YH, Li MY. Changes in transferrin and hepcidin genes expression in the liver of the fish *Pseudosciaena crocea* following exposure to cadmium. Arch Toxicol 2008;82:525-30.

Chuu JJ, Huang ZN, Yu HH, Chang LH, Lin-Shiau SY. Attenuation by methyl mercury and mercuric sulfide of pentobarbital induced hypnotic tolerance in mice through inhibition of ATPase activities and nitric oxide production in cerebral cortex. Arch Toxicol 2008;82:343-53.

Do Amaral CL, Francescato HD, Coimbra TM, Costa RS, Darin JD, Antunes LM, Bianchi Mde L. Resveratrol attenuates cisplatin-induced nephrotoxicity in rats. Arch Toxicol 2008;82:363-70.

Glahn F, Schmidt-Heck W, Zellmer S, Guthke R, Wiese J, Golka K, Hergenröder R, Degen GH, Lehmann T, Hermes M, Schormann W, Brulport M, Bauer A, Bedawy E, Gebhardt R, Hengstler JG, Foth H. Cadmium, cobalt and lead cause stress response, cell cycle deregulation and increased steroid as well as xenobiotic metabolism in primary normal human bronchial epithelial cells which is coordinated by at least nine transcription factors. Arch Toxicol 2008;82:513-24.

Kanda H, Sumi D, Endo A, Toyama T, Chen CL, Kikushima M, Kumagai Y. Reduction of arginase I activity and manganese levels in the liver during exposure of rats to methylmercury: a possible mechanism. *Arch Toxicol* 2008a;82:803-8.

Kanda H, Kikushima M, Homma-Takeda S, Sumi D, Endo A, Toyama T, Miura N, Naganuma A, Kumagai Y. Downregulation of arginase II and renal apoptosis by inorganic mercury: overexpression of arginase II reduces its apoptosis. *Arch Toxicol* 2008b; 82:67-73.

Kobayashi Y, Negishi T, Mizumura A, Watanabe T, Hirano S. Distribution and excretion of arsenic in cynomolgus monkey following repeated administration of diphenylarsinic acid. *Arch Toxicol* 2008;82: 553-61.

Krieg EF Jr, Chrislip DW, Brightwell WS. A meta-analysis of studies investigating the effects of lead exposure on nerve conduction. *Arch Toxicol* 2008;82:531-42.

Liao Y, Yu F, Jin Y, Lu C, Li G, Zhi X, An L, Yang J. Selection of micronutrients used along with DMSA in the treatment of moderately lead intoxicated mice. *Arch Toxicol* 2008;82:37-43.

Manna P, Sinha M, Sil PC. Arsenic-induced oxidative myocardial injury: protective role of arjunolic acid. *Arch Toxicol* 2008;82: 137-49.

Müller YM, Rivero LB, Carvalho MC, Kobus K, Farina M, Nazari EM. Behavioral impairments related to lead-induced developmental neurotoxicity in chicks. *Arch Toxicol* 2008;82:445-51.

Naraharisetti SB, Aggarwal M, Sarkar SN, Malik JK. Concurrent subacute exposure to arsenic through drinking water and malathion via diet in male rats: effects on hepatic drug-metabolizing enzymes. *Arch Toxicol* 2008;82:543-51.

Rezende VB, Barbosa F Jr, Montenegro MF, Sandrim VC, Gerlach RF, Tanus-Santos JE. Haplotypes of vitamin D receptor modulate the circulating levels of lead in exposed subjects. *Arch Toxicol* 2008;82: 29-36.

Verstraeten SV, Aimo L, Oteiza PI. Aluminium and lead: molecular mechanisms of brain toxicity. *Arch Toxicol* 2008;82:789-802.

Yu F, Liao Y, Jin Y, Zhao Y, Ren Y, Lu C, Li G, Li Y, Yang J. Effects of in utero meso-2,3-dimercaptosuccinic acid with calcium and ascorbic acid on lead-induced fetal development. *Arch Toxicol* 2008;82: 453-9.